Modelling signalling pathways in cancer growth

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Several signalling pathways control normal cell processes like cell proliferation, division, cellular adhesion, migration and apoptosis, with calcium (Ca^{2+}) signalling being one of the most critical. While these processes are essential in maintaining order in organisms, they can become faulty in malignant cells, promoting cancer growth. They have thus generated a lot of interest over the past years. In particular, cellular adhesion and proliferation are fundamental features of multicellular organisms, and they play an important role in cancer. Cellular adhesion is mediated through cell-adhesion molecules (CAMs), two major families of which are integrins and cadherins. Here we present models consisting of a nonlocal partial differential equation for the cancer cell density coupled with nonlinear differential equations modelling important chemical pathways, mainly paying attention to Ca^{2+} signalling; in both models the cell density equation includes cancer cell proliferation and cellular adhesion, which is described by a non-local term. As Ca^{2+} signalling presents diverse behaviour as the IP_3 level increases, such as solitary waves and periodic wave-trains, we investigate the interplay of Ca^{2+} signals with the cancer cell density. We study a series of scenarios, varying the adhesion effect, and determine the speed of cancer cell movement and aggregation. *This project has been carried out in collaboration with K. Kaouri and R. Thul, and has received funding from the European Union's H2020 Research and Innovation Action under Grant Agreement No 741657 (SciShops.eu).*